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Cerebral, cardiac and skeletal muscle stress associated with a series of static and dynamic apnoeas

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Abstract

Purpose-This study sought to explore, for the first time, the effects of repeated maximal static and dynamic apnoeic attempts on the physiological milieu by assessing cerebral, cardiac and striatal muscle stress-related biomarkers in a group of elite breath-hold divers (EBHD).

Methods-Sixteen healthy males were recruited (EBHD=8; controls=8). On two separate occasions EBHD performed two sets of five repeated maximal static apnoeas (STA) or five repeated maximal dynamic apnoeas (DYN). Controls performed a static eupnoeic protocol to negate any effects of water immersion and diurnal variation on haematology (CTL). Venous blood samples were drawn at 30, 90, and 180-mins after each protocol to determine S100β, neuron-specific enolase (NSE), myoglobin and high sensitivity cardiac troponin T (hscTNT) concentrations.

Results-S100β and myoglobin concentrations were elevated following both apnoeic interventions ($p<0.001$; $p\leq0.028$, respectively) but not after CTL ($p\geq0.348$). S100β increased from baseline (0.024±0.005µg/L) at 30 (STA, +149%, $p<0.001$; DYN, +166%, $p<0.001$) and 90 mins (STA, +129%, $p<0.001$; DYN, +132%, $p=0.008$) following the last apnoeic repetition. Myoglobin was higher than baseline (22.3±2.7ng/mL) at 30 (+42%, $p=0.04$), 90 (+64%, $p<0.001$) and 180 mins (+49%, $p=0.013$) post-STA and at 90 mins (+63%, $p=0.016$) post-DYN. Post-apnoeic S100β and myoglobin concentrations were higher than CTL (STA, $p<0.001$; DYN, $p\leq0.004$). NSE and hscTNT did not change

from basal concentrations after the apnoeic ($p \geq 0.146$) nor following the eupnoeic ($p \geq 0.553$) intervention.

Conclusions-This study suggests that a series of repeated maximal static and dynamic apnoeas transiently disrupt the blood-brain barrier and instigate muscle injury but do not induce neuronal-parenchymal damage or myocardial damage.

Keywords: Hypoxaemia, Breath-hold, Diving, Myoglobin, Neuron-specific Enolase, S100β, Cardiac troponin, Apnoea

Introduction

Breath-hold diving continues to gain popularity and recognition as both a competitive and recreational sport. The continued progression of world records is astonishing, particularly given the extreme hypoxaemic hypercapnia and hydrostatic pressures these athletes endure; yet the continued pursuit of performance raises safety concerns [1]. To date, a breadth of research exists that has delineated the physiological characteristics of breath-hold divers, as well as the responses that occur during and/or following prolonged apnoeic bouts (e.g., the diving response, trigeminocardiac reflex, splenic contractions, erythropoietic responses, etc.) [1-8]. In contrast, there is a paucity of literature concerning the possible health implications associated with exposure to such activities [9-14].

Emerging evidence indicates that a single, maximal static apnoeic attempt is capable of transiently disrupting the blood-brain barrier [9, 13] and instigates neuronal-parenchymal damage [11] but is not associated with cardiac injury [10, 15]. Specifically, both Andersson et al. [9] and Bain et al. [13] reported significant increases in serum S100 calcium-binding protein β (S100β) at the end of a maximal static apnoea (~26%, 335 ± 38 s; ~40%, 307 ± 64 s, respectively), which is indicative of a potential perturbation of the blood-brain barrier. Similarly, in competitive breath-hold divers ~1 h following a single maximal static apnoeic attempt (mean ~5 min [range: 2.3–7.6 min]), total tau and amyloid β42 concentrations were significantly increased from basal concentrations [11]. Furthermore, neuron-specific enolase (NSE),

a prognostic indicator of traumatic brain injury [16], was shown to be significantly elevated (~70%) ~3 h following a combined bout of static and dynamic apnoeas [12]. Taken together, these findings highlight the severity of physiological stress encountered by breath-hold divers during maximal apnoeic activity.

To date, only one study has examined the physiological stress imposed by repeated apnoeic exposures on bodily organs (e.g., the heart) [15]. Specifically, following repeated apnoeic dives (i.e., performed over a 5 h spearfishing competition) cardiac troponin I increased (+275%) as did brain natriuretic peptide (+229%), which is notable as both are markers of cardiac stress [15, 17]. These increases were not evident following a single maximal static apnoeic attempt [15], nor after a combined bout of static and dynamic apnoeas [12]. However, to the best of our knowledge, no study has investigated the magnitude of physiological stress instigated by a series of repeated maximal static and/or dynamic apnoeic bouts on cerebral, cardiac, and striatal muscle markers. Contrary to apnoeic competitions (i.e., with the exception of spearfishing competitions), whereby athletes perform one maximal attempt per discipline; during training and spearfishing, breath-hold divers commonly perform a series of repeated sub-maximal and/or maximal attempts. Intermittent hypoxaemia of this nature is capable of upregulating the production of reactive oxygen species [18, 19], exacerbating oxidative stress levels [20, 21] as well as inflicting oxidative damage on cells, tissues and organs. Collectively, these observations reinforce the necessity for further studies to be undertaken to assess the possible health consequences associated with repeated apnoeic exposures.

Duly, this study sought to explore the effects of repeated maximal static and dynamic apnoeic attempts on the physiological milieu by assessing cerebral (NSE and S100β), cardiac (cardiac troponin T) and striatal muscle (myoglobin) stress-related biomarkers in a group of elite breath-hold divers (EBHD). We hypothesised that both repeated maximal apnoeic modalities would be associated with significant physiological stress.

Materials and Methods

Participants

Sixteen healthy males volunteered for this study and were differentiated into two groups: EBHD (n = 8) and controls (n = 8). All breath-hold divers actively participated in national and/or international competitions (Table 1) and the control group comprised of physically active individuals. Participants were non-smoking, habitual sea-level residents and provided written informed consent before the study. The study received institutional ethical approval and all experimental procedures were completed in accordance with the Declaration of Helsinki. The study was part of a larger project investigating physiological responses to a series of repeated apnoeas [5].

Experimental Protocol

During each testing session participants reported to the laboratory at Leeds Beckett University (Leeds, England) after a 12 h fast and refrained from consuming caffeine- and alcohol-containing beverages. Additionally, participants were instructed to avoid physical activity and apnoea-related activities for 24 h prior to and during each testing day.

Table 1 about here

Preliminary Resting Measurements

Following arrival at the laboratory the participants' height and body mass were assessed (Seca, Vogel & Halke, Germany) (Table 1). Thereafter, the participants underwent a 20-mins supine resting period and subsequently, two venous blood samples (5 mL) were drawn from a median cubital or basilic vein to assess for resting serum NSE, S100β, myoglobin and high sensitivity cardiac troponin T (hscTNT) concentrations (BD Vacutainer, 367954, UK).

Familiarisation Session

Participants underwent a familiarisation session within 24 h of completing the baseline measurements to introduce them to the testing environment, trial conditions and requirements.

Figure 1 about here

Apnoeic Protocols

Within a week from completing the familiarisation session, participants reported to the swimming pool (~28 °C) facilities and, under the supervision of a qualified safety diver(s) performed, on separate days (i.e., separated by ≥96 h), in a Latin-Square fashion and randomised order (achieved using a computer-generated list of random numbers [https://www.randomizer.org/]) one of the following protocols: five repeated maximal dynamic apnoeas without fins (horizontal underwater swimming), or two sets of five maximal static apnoeic attempts (i.e., two sets separated by a 10-mins seated rest; breath-holding performed in a prone/semi-seated position on the water surface) (Figure 1). To control for diurnal oscillations of the measured variables both apnoeic protocols were performed at the same time of the day.

Participants were instructed to hold their breath after a deep but not maximal inspiration, without prior hyperventilation or glossopharyngeal insufflation. After each maximal attempt, participants underwent a two-minute resting period whereby they were allowed to relax and breathe normally in a seated position, whilst remaining immersed in water up to waist height (Figure 1). This procedure was repeated five times per set and performance data (i.e., apnoeic duration and/or distance covered) were recorded during each maximal attempt (Figure 1).

Control Protocol

To control any possible effects of whole-body immersion in water, a control group performed a static eupnoeic (normal breathing) protocol. The static eupnoeic protocol replicated the water exposure times, resting periods and data

collection timepoints of the static apnoea protocol (since the water exposures were longer in the static versus the dynamic apnoea protocol) and replaced apnoeas with normal breathing periods.

Participants reported to the swimming pool facilities at the same time of day as during the apnoeic protocols and were immersed in water up to the neck.

Post-Apnoea Blood Sample

At completion of the apnoeic and control protocols, a cannula was inserted into a suitable median cubital or basilic vein of the participant's arm and two venous blood samples were drawn at 30, 90 and 180 mins after the last apnoeic/eupnoeic repetition to determine the serum concentrations of circulating NSE, S100β, myoglobin and hscTNT.

Blood Sample Treatment

Samples were gently inverted, allowed to coagulate at room temperature for 20 mins then centrifuged (4000 rpm for 10 mins at 4°C; ALC Multispeed Refrigerated Centrifuge, PK131R, UK) and the serum supernatants were frozen at -80°C for subsequent analyses.

Blood Analyses

Enzyme-linked immunosorbent assay (ELISA) was performed to assess serum concentrations of NSE (R&D Systems, Quantikine IVD ELISA, Human Enolase 2/Neuron-specific Enolase Immunoassay, DENL20, sensitivity 0.038 ng/mL; intra-assay variability ~2.2%), S100β (R&D Systems, Human S100β Duo Set ELISA, DY1820; intra-assay variability ~4.5%) and myoglobin (Abcam, Human Myoglobin ELISA, ab108652, sensitivity 5 ng/mL; intra-assay variability ~5.4%). hscTNT was quantified by electro-chemiluminescence immunoassay (Cobas Analyser, Roche Diagnostics) (intra-assay variability ~6%).

Statistical Analysis

All participants completed the protocols successfully and all data were statistically analysed using the IBM SPSS statistics software (Version 21, Armonk, NY). The Shapiro-Wilk test was used to assess normality, whereas homogeneity was assessed using Levene's test. Sphericity was evaluated using Mauchly's test of sphericity; for instances where the assumption of sphericity was violated, the Greenhouse-Geisser correction was applied. Repeated measures ANOVAs with Tukey's post-hoc tests were used to assess within-group differences for baseline measurements and other timepoints for serum S100β, NSE, myoglobin, and hscTNT concentrations. Two-way ANOVAs were used to assess temporal differences between conditions. Data are reported as mean ± SD, with significance accepted at $p < 0.05$. Figures were constructed using GraphPad Prism (GraphPad Software, Version 7.0c, La Jolla, California, USA).

Results

Apnoeic Performances

Mean static apnoea duration was 218 s (range 130-350 s) and mean dynamic apnoea distance covered without fins was 71 m (range 46-126 m).

S100β

Mean post-apnoeic S100β concentrations were significantly higher than baseline (0.024 ± 0.005 µg/L) following the static ($p < 0.001$) and dynamic ($p < 0.001$) apnoea protocols, while no differences were observed during the control protocol ($p = 0.348$) (Figure 2a). Notably, S100β concentrations were greater than baseline at 30 (static, +149%, 0.059 ± 0.019 µg/L, $p < 0.001$; dynamic, +166%, 0.061 ± 0.019 µg/L, $p < 0.001$) and 90 mins (static, +129%, 0.055 ± 0.019 µg/L, $p < 0.001$; dynamic, +132%, 0.054 ± 0.018 µg/L, $p = 0.008$) after the last apnoeic repetition but not at 180 mins (static, +32%, 0.031 ± 0.008 µg/L, $p = 0.676$; dynamic, +46%, 0.035 ± 0.011 µg/L, $p = 0.432$). In addition, mean post-apnoeic S100β concentrations were significantly higher than control (static, $p < 0.001$; dynamic, $p = 0.002$) at 30 ($p < 0.001$), 90 ($p < 0.001$) and 180 mins ($p \leq 0.006$), whereas no differences were documented between the apnoeic protocols ($p = 0.622$) (Figure 2a).

Neuron-Specific Enolase

There was no effect of the apnoeic (static, $p = 0.146$; dynamic, $p = 0.836$) nor eupnoeic ($p = 0.988$) interventions on serum NSE concentrations compared with baseline (EBHD, 2.91 ± 0.55 pg/mL; control, 3.32 ± 0.63 pg/mL). Moreover, there were no between-protocol differences (static *vs.* dynamic, $p = 0.682$; apnoeic *vs.* control, $p \geq 0.127$) (Figure 2b).

High Sensitivity Cardiac Troponin T

hscTNT concentrations were not significantly different from baseline (EBHD, 5 ± 1 ng/L; control, 6 ± 1 ng/L) neither after the apnoeic interventions ($p \geq 0.224$) nor following the control protocol (6 ± 1 ng/L, $p = 0.553$) (Figure 2c). In addition, there were no between-protocol differences (static *vs.* dynamic, $p = 0.384$; apnoeic *vs.* control, $p \geq 0.163$) (Figure 2c).

*****Figure 2 about here*****

Myoglobin

There was a significant increase in myoglobin concentrations after both the static ($p < 0.001$) and dynamic ($p = 0.028$) apnoea protocols, whereas no significant differences were denoted during control ($p = 0.493$) (Figure 3a). Specifically, myoglobin was significantly higher than baseline (22.3 ± 2.7 ng/mL) at 30 (+42%, 31.2 ± 3.3 ng/mL, $p = 0.040$), 90 (+64%, 36.5 ± 11.5 ng/mL, $p < 0.001$) and 180 mins (+49%, 32.7 ± 7.3 ng/mL, $p = 0.013$) following the last static apnoeic bout (Figure 3a), whereas during the dynamic apnoea intervention myoglobin concentrations were

only elevated at 90 mins (+63%, 35.5 ± 16.7 ng/mL, p = 0.016) after the last apnoeic repetition.

Figure 3 about here

Resting baseline concentrations were significantly higher in the control group (EBHD, 22.3 ± 2.7 ng/mL; control, 27.5 ± 7.2 ng/mL, p = 0.047) (Figure 3a), as such, between-group differences (EBHD *vs*. control) were compared using delta percentage change (Figure 3b). Myoglobin concentrations were significantly higher in response to the static (p < 0.001) and dynamic apnoea (p = 0.004) protocols versus control, with no differences observed between the apnoeic protocols (p = 0.587) (Figure 3b).

Discussion

This study examined the effects of repeated maximal apnoeic bouts on the physiological milieu by assessing cerebral, cardiac and skeletal muscle stress-related biomarkers. The primary findings demonstrate that a series of repeated maximal static and dynamic apnoeas incite a significant rise in S100β and myoglobin concentrations without any detectable changes in NSE nor hscTNT. Taken together, our study suggests that a series of repeated maximal apnoeic attempts induce muscle injury and signify a potential, albeit minor blood-brain barrier disruption that appears to occur in the absence of neuronal-parenchymal damage.

S100β was significantly elevated from baseline only following the apnoeic interventions (Figure 2a). S100β is a dimeric calcium-binding protein and is predominantly found in brain astrocytes [22]. Molecular communication between blood and the brain is largely prevented by the blood-brain barrier, a highly selective semipermeable border that is primarily composed of microvascular endothelial cells linked by tight and adherent junctions [22, 23]. Evidence suggests that astrocytic proteins extravasate into the serum only when the blood-brain barrier is breached [24], with a direct correlation reported between the venous concentration of S100β and the magnitude of the blood-brain barrier opening [25]. As such, our study suggests that a series of repeated maximal apnoeas is capable of transiently disrupting the blood-brain barrier. Considering that S100β exerts both neurotrophic and gliotrophic roles, it is presently unclear whether these increases serve a neuroprotective purpose (reactive astrogliosis) or rather represent a more menacing phenomenon (e.g., glial damage) [26].

In spite of never reaching pathological limits (> 0.10 µg/L) [27], the transient S100β increases documented in the present study are higher than those previously reported in competitive breath-hold divers following a single, dry maximal static apnoeic attempt [9, 13] and after a combined bout of static and dynamic apnoeas [12]. It is currently well accepted that intermittent hypoxaemia has a dose-dependent association (i.e., severity of hypoxaemia, duration of exposure) with an increase in the permeability of the blood-brain barrier [23]. This response is believed to be orchestrated, at least in part, by reactive oxygen species [28, 29]. Interestingly, using a contrast-enhanced magnetic resonance imaging technique, Kanner et al. [25] observed a direct correlation between S100β concentrations and the magnitude of the opening of the blood-brain barrier. It is, therefore, tempting to speculate that the higher post-apnoeic S100β concentrations recorded in the present study may relate to the nature of our experimental design which comprised of a series of repeated episodes of hypoxaemia interspersed with short periods of normal breathing.

It is generally accepted in the existing literature that S100β is a peripheral biochemical marker that is implicated in brain damage and neurodegenerative processes [22]. It is also noteworthy that, in contrast to NSE, S100β is expressed in skeletal muscle myofibers and is locally released in response to myofiber damage and degeneration [30, 31]. Moreover, S100β appears to be acutely important for skeletal muscle regeneration, due to its effects on myoblast proliferation through stimulation of extracellular signal-regulated kinase 1/2 [32]. Indeed, neutralization of S100β release from acutely injured wild-type skeletal muscles reduced the population expansion of activated satellite cells, lowered the infiltration of injured tissues with macrophages as well as delayed the transition of macrophages from the M1 (proinflammatory) to the M2 (anti-inflammatory and pro-regenerative) phase [31, 32]; conjointly impairing the regenerative process. However, persistently high S100β levels may compromise the regenerative process by blocking myogenic differentiation [31] through inhibition of p38 mitogen-activated protein kinase [31, 32]. Therefore, considering also the lack of changes in serum NSE, an alternative explanation for our findings might be that the elevated serum S100β levels may also stem from injured myofibers and represent a transient response to support the regeneration of injured skeletal muscle, by increasing the myoblast population in the local tissue area and preventing precocious myoblast differentiation.

A series of repeated maximal apnoeas did not elicit any changes in circulating NSE concentrations (Figure 2b), attesting against any form of neuronal-parenchymal damage [16, 22]. Our findings are in contrast to those of Kjeld et al.[12], whereby a significant rise in plasma NSE (from 14.5 ± 5.3 ng/mL to 24.6 ± 6.4 ng/mL) was documented ~3 h post (i.e., a range of post-apnoea timepoints 94-257 mins) a combined maximal bout of static and dynamic apnoeas. It is noteworthy, however, that in the Kjeld et al.[12] study, nine out of 17 breath-hold divers suffered a blackout episode (loss of consciousness) during their maximal attempts. Liner and Andersson [33] demonstrated that a blackout episode is associated with disruption of the blood-brain barrier as evidenced by a significant rise in serum S100β; increases that persisted for more than a day following the blackout incident. Hence, it is perhaps unsurprising that increases in NSE have been reported by Kjeld et al. [12]. Contrastingly, in our study, none of our participants suffered a blackout nor exhibited any signs associated with loss of motor control (e.g., confusion, postural disturbance, spasms, speech problems, unresponsiveness, etc.). Taken together, our findings suggest that in the absence of a hypoxaemic syncope, a series of repeated maximal apnoeas does not incite neuronal-parenchymal damage.

Myoglobin, a marker of muscle injury, was markedly elevated following both apnoeic interventions, while no changes were detected in the control group (Figure 3). Our findings align with those of Marlinge et al. [15] who documented a similar rise in myoglobin following a 5 h spearfishing competition. These increases were not evident following a single maximal static apnoeic bout either with [10], or without glossopharyngeal insufflation [15]. Repeated maximal apnoeas have been linked with the upregulation of reactive oxygen species production [18] and aggravation of oxidative stress levels [20]; undulations that, in the absence of sufficient antioxidant enzyme defences, are associated with oxidative damage[19]. Specifically, excessive free radical accumulation instigates muscle cell damage, resulting in dysregulation of sodium-calcium channel functioning, ultimately elevating intracellular free ionized calcium [34]. This causes a resultant activation of calcium-dependent enzymes, which go on to further metabolise and rupture the sarcolemma [34]. Consequently, intracellular contents such as myoglobin and creatine kinase are released into the

circulation [34]. Therefore, our study demonstrates that, in a similar manner to repeated apnoeic dives, a series of repeated maximal static and dynamic apnoeas are associated with striatal muscle injury.

The presently recorded myoglobin increases are comparable to those reported following a 90-min cycling exercise bout (power output held at 90W) [35] and those after eccentric-concentric exercise [36], but are substantially lower than those documented following a maximal endurance exercise bout performed under normoxic and hypoxic conditions [37]. Collectively, the magnitude of myoglobin release denoted following a series of repeated maximal apnoeic epochs is well within the physiological limits (> 85 ng/ml), hence, our findings suggest that, at least in EBHD, the risk of sustaining rhabdomyolysis is very low [38]. However, what might seem surprising is that the post-apnoeic myoglobin concentrations did not differ across our apnoeic interventions (Figure 3), despite significantly lower end-apnoeic SpO_2 levels attained during the dynamic protocol (62 ± 10 % *vs.* 76 ± 5 %) [5]. It is possible that the greater number of apnoeic repetitions incorporated in our static apnoea protocol (i.e., 10 *vs.* 5 repetitions) may have exacerbated the production of reactive oxygen species, consequently aggravating oxidative damage and promoting a similar release of myoglobin. A measure of oxidative stress would have certainly provided additional insights to the mechanistic basis of this effect.

To evaluate the magnitude of the cardiovascular burden imposed by repeated maximal apnoeic bouts we assessed hscTNT (Figure 2c), a regulatory protein that is expressed in cardiac myocytes and serves as a specific biomarker of myocardial injury [17]. Interestingly, post-apnoeic cardiac troponin T concentrations did not differ from baseline, suggesting that a series of repeated maximal apnoeic bouts does not incite myocardial injury. Our data align well with earlier studies that showed no changes in cardiac troponin following a static apnoea packing-blackout that included episodes of asystole (cardiac troponin T [10]), a single dry maximal static apnoeic attempt (cardiac troponin I [15]), or a combined bout of static and dynamic apnoeas (hscTNT [12]). They also concur with longitudinal studies that did not unveil any cardiac abnormalities nor morphological alternations in EBHD [1, 39]. Our findings do, however, contrast with those of Eichhorn et al. [14] who documented a significant rise in hscTNT 4 h following a single, maximal, dry static apnoeic attempt in a group of EBHD. It is noteworthy that this increase was lower [(pre) 2.2 ± 1.1 pg/ml *vs.* (post) 3.1 ± 1.7 pg/ml] than that recorded in the present study [(pre) 5.3 ± 0.5 ng/L *vs.* (3 h post) static 7 ± 1.6 ng/L and dynamic 6.9 ± 2.2 ng/L]. Thus, the current study signifies that a series of repeated maximal apnoeic attempts does not evoke myocardial damage.

To conclude, this study suggests that a series of repeated maximal static and dynamic apnoeas are associated with a potential, albeit minor, transient disruption of the blood-brain barrier as evidenced by a rise in S100β and instigate muscle injury as evinced by a rise in myoglobin but do not cause any detectable neuronal-parenchymal damage nor myocardial damage.

Perspective

Considering the growing popularity of breath-hold diving as a competitive and recreational sport, enhancing our understanding of the possible health implications associated with exposure to such activities is paramount from a

safety and medical standpoint[1, 40]. In this context, the present study demonstrates that, in EBHD, a series of repeated maximal apnoeic bouts do not incite any detectable neuronal-parenchymal damage nor myocardial damage but are associated with a potential transient, albeit minor, disruption of the blood-brain barrier and muscle injury. It is presently unclear whether these physiological responses are coherently expressed in non-divers. As such, it is imperative that further research is conducted to evaluate the possible health risks associated with apnoeic training.

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Table Legends

Table 1. Mean (± standard deviation [SD]) participant characteristics.

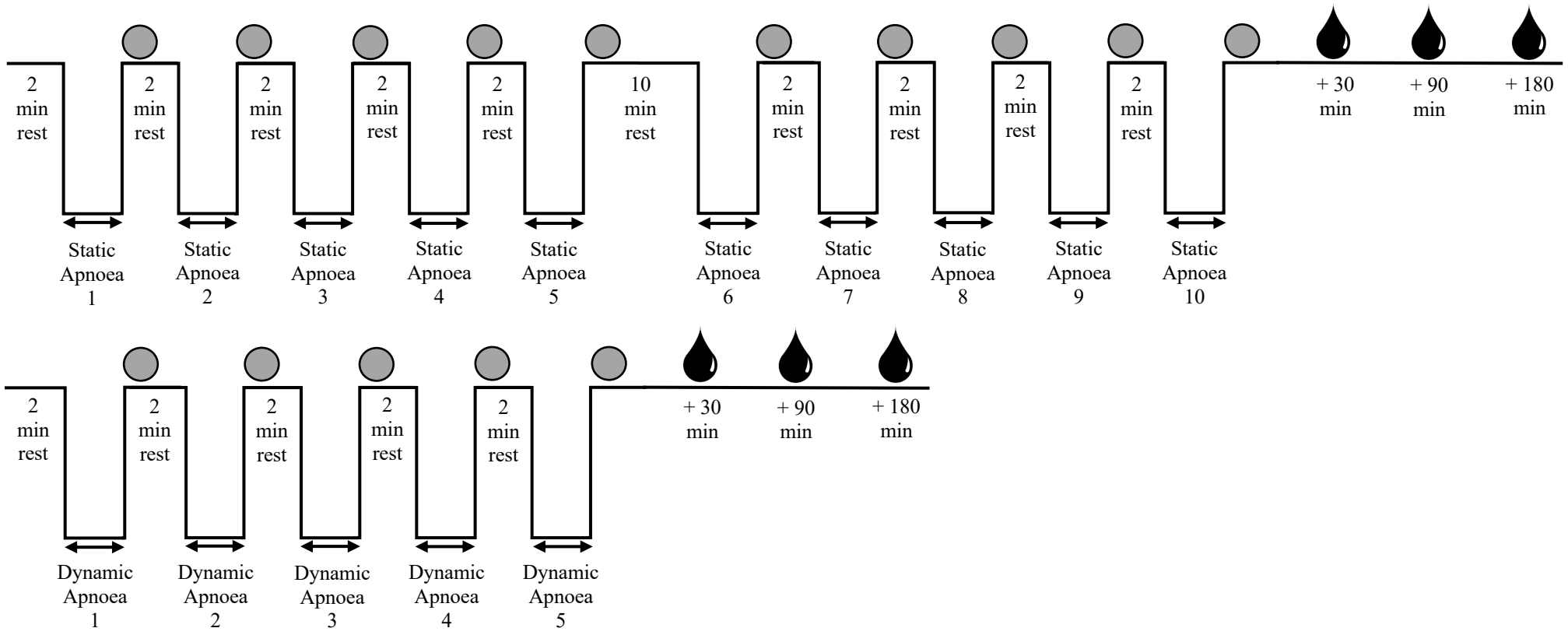
Figure Legends

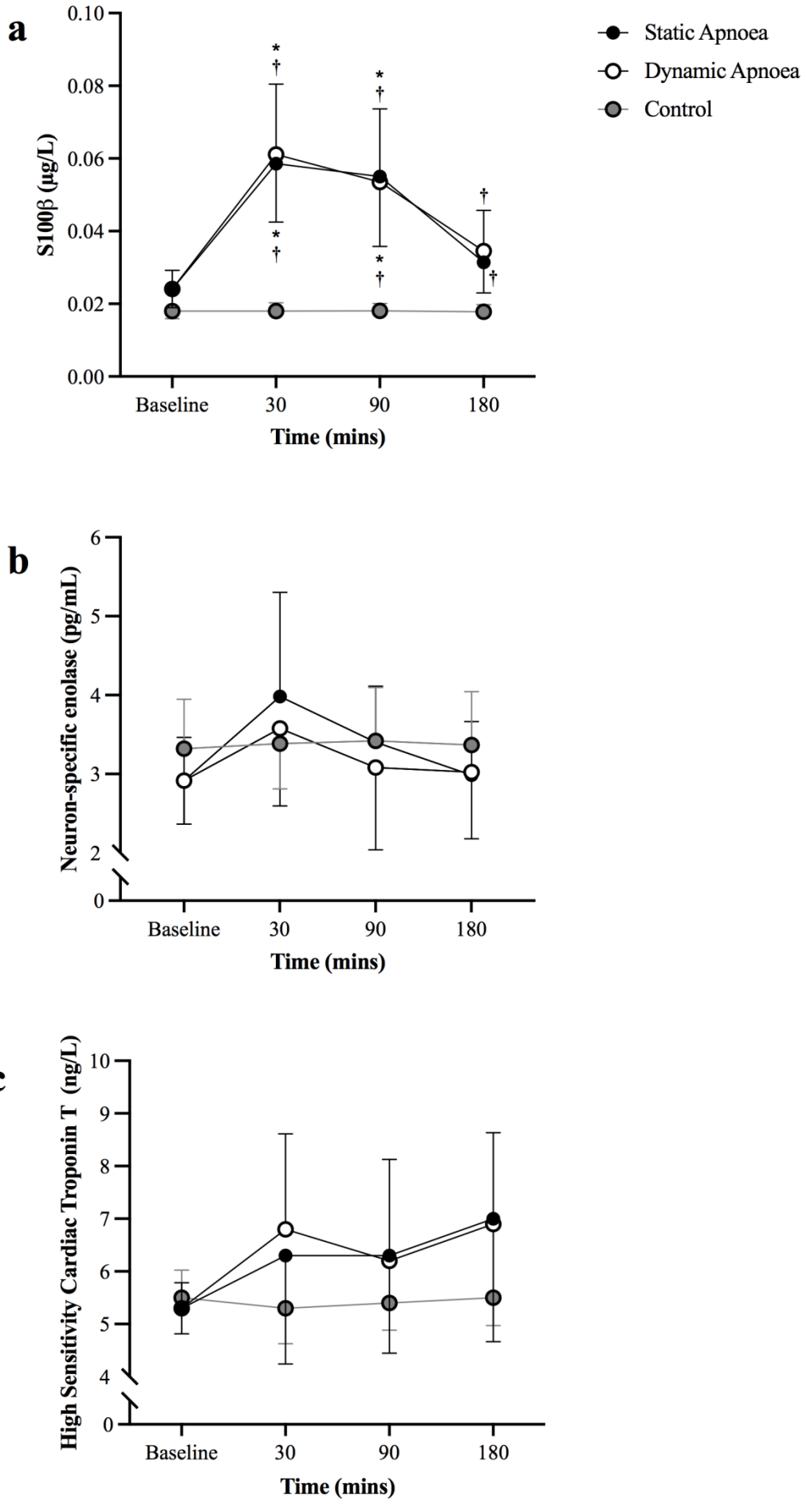
Figure 1. Schematic representation depicting the data collection time points during the static (a) and dynamic (b) apnoea protocols.

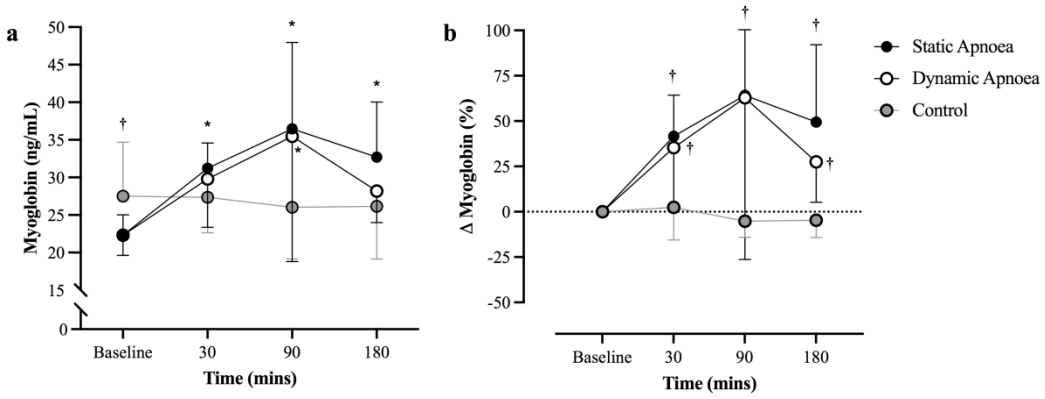
Figure 2. Mean S100β (a), NSE (b) and hscTNT (c) concentrations from baseline to 180 mins after the apnoeic and eupnoeic protocols. Data are presented as mean ± SD. * denotes significant difference ($p < 0.05$) compared to baseline; † represents significant difference between apnoeic and eupnoeic protocols. S100β, S100 calcium-binding protein β; NSE, neuron-specific enolase; hscTNT, high sensitivity cardiac troponin T; µg/L, microgram per litre; pg/mL, picograms per millilitre; ng/L, nanograms per litre.

Figure 3. Absolute (a) and delta percentage change (b) in mean myoglobin from baseline to 180 mins after the apnoeic and eupnoeic protocols. Data are presented as mean ± SD. * denotes significant difference ($p < 0.05$) compared to baseline; † represents significant difference between apnoeic and eupnoeic protocols. ng/mL, nanograms per millilitre.

Variables	EBHD (*n* = 8)	Control (*n* = 8)
Age (yrs)	39 ± 7	28 ± 5
Height (m)	1.8 ± 0.1	1.8 ± 0.1
Body mass (kg)	84 ± 12	82 ± 11
Body mass index (kg/m^2)	26 ± 1.3	25 ± 2.2
Years practicing apnoea (yrs)	7 ± 2	-
Personal best static apnoea (s)	376 ± 39	-
Personal best dynamic apnoea with fins (m)	193 ± 42	-
Personal best dynamic apnoea without fins (m)	131 ± 41	-







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